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Latin America: Should We Be Worried about Zika Virus?

About the Author: Joselyn Ye is currently a fifth-year medical student at Cayetano Heredia University, Lima, Peru of a seven-year program.

To the Editor,

The Zika virus (ZIKV) was identified for the first time in 1947, almost 70 years ago. Despite being known during all these decades, it has been neglected until the current outbreak in Brazil. This is an emergent disease that has been developing slowly throughout these years; we still have limited information and scientific knowledge about this illness and its potential outcomes.¹

This infection causes fairly mild effects, as 80% of the patients go unnoticed.² The most common symptoms are fever, rash, joint pain, and conjunctivitis, which last for several days to a week after the infection.³ The primary mode of transmission is through the bite of an infected mosquito of the *Aedes* species. Apart from mosquitoes, other non-vector means of transmission are sexual intercourse and during pregnancy. Furthermore, there is a strong possibility that ZIKV can be spread through blood transfusions. It is suggested that breastfeeding could be another way of transmission, due to the fact that it has been found in breast milk. However, there are no reports up to date about other means of transmission. Because of the benefits of breastfeeding, mothers are encouraged to breastfeed even in areas where ZIKV is found (Available from: <http://www.cdc.gov/zika/about/overview.html>; cited 2016 Jul 28).

Since ZIKV can cross the blood brain barrier,⁴ a relationship with some severe congenital neurologic defects, such as mi-

crocephaly and Guillain-Barré syndrome, has been established. Mlakar et al described that the cases of microcephaly increased by a factor of 20 among newborns in the northeast of Brazil.^{4,5} Many other authors also describe severe brain injuries, inflammatory signs and calcifications, leading them to think that it is likely that an intrauterine infection brings about the abnormal brain development, and can lead to cerebral palsy after birth.¹⁻³ It is also suggested that the low maternal supplementation of nutrients may affect other organs as well,⁶ although this is yet to be determined.

The World Health Organization declared ZIKV a 'Public Health Emergency of International Concern';⁷ as it threatens to become a global pandemic. Up to date, there is no cure or vaccine available (Available from: <http://www.cdc.gov/zika/about/overview.html>; cited 2016 Jul 28). There are different methods for diagnosing ZIKV. One of them is the real-time reverse transcription-polymerase chain reaction (rRT-PCR), which must only be conducted on urine or serum samples collected during the first 14 days after symptoms onset. This test has a high sensitivity and a poor specificity. If the rRT-PCR is negative in a high risk patient, the ELISA for qualitative detection of IgM (Zika MAC-ELISA) is used.⁸ However, a cross-reaction with other flaviviruses is present, as such the test has poor specificity in people living in endemic areas that have been previously exposed to the dengue and yellow fever viruses.⁶⁻⁹ Therefore, results may be difficult to interpret in Latin America.

System-wide barriers impede health care delivery in our region. Latin American health providers have the obligation to generate awareness amongst our people about ZIKV. The need for a stronger surveillance system, suitable diagnostics methods, communication about ZIKV outbreak risks, vector control and adequate guidance to pregnant women is necessary.³

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Methylprednisolone-Responsive Leptospiral Acute Pulmonary Syndrome

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To the Editor,

The article by Johan-Arief et al highlights the role of early methylprednisolone therapy to reduce morbidity and mortality of the acute pulmonary syndrome in leptospirosis,¹ and the authors emphasize the ideal administration of this drug within 12 hours after diagnosis. They reported a 26-year-old man with fever of one-week duration, who on the third day had presented with cough and breathlessness, and episodes of hemoptysis occurred two days later.¹ His professional work involved frequent close contact with soil, and he was swimming in a waterfall a week before the onset of the high-grade fever associated with chills, rigors and body aches.¹ Physical signs were jaundice, subconjunctival suffusion, and bilateral pulmonary crepitations. Blood tests showed neutrophilic leucocytosis, low platelets, disordered coagulation, liver and renal functions; and the chest x-ray revealed scattered alveolar infiltrates. The Weil's disease was diagnosed by positive IgM ELISA, and the specific microscopic agglutination test (1:400).¹ Intravenously, he underwent tranexamic acid (500 mg 3 times daily), plus benzyl penicillin (1200 mg every 6 hours) during a week, and methylprednisolone (15 mg/kg daily) for 3 days.¹ His clinical response to treatment was good and he was soon discharged home asymptomatic. Environmental factors and typical features of severe leptospirosis raised the diagnostic suspicion; as the early diagnosis is the cornerstone of prompt adequate treatment of severe leptospirosis, one must consider some main concerns that constitute frequent challenges in clinical practice.^{2,3} During the first week of acute leptospirosis, the blood borne bacteria almost invariably do not evoke an immune response enough to yield antibody levels and false negative tests can occur;¹⁻³ so, the goal of starting adequate therapy within the first seven days of disease may be hindered.³ Pulmonary involvement with hemoptysis has been more often reported in severe Weil's disease, and the coexistence of accentuated thrombocytopenia may play a role on the poorest prognoses. Significant pulmonary changes as well as low platelet counts can pose other concerns about diagnostic pitfalls with Hantavirus and dengue

infections, especially in developing countries.²⁻⁵ Since the first reports in North American Indians, the possibility of mistakes involving the acute pulmonary syndrome caused by leptospirosis and HPS was commented in Brazilian Journals.⁴ Brazilian authors described a 19-year-old man with the diagnosis of anicteric leptospirosis, who had a productive cough without hemoptysis, reduced lung sounds on the right lower third of the thorax, and normal kidney function. Images of computed tomography confirmed the acute inflammatory process in the right lung, and he underwent a successful course of intravenous ceftriaxone.³ Worthy of note, the patient had clinical and electrocardiographic findings of pericarditis; moreover, the count of bands was high (up to 1020) and the platelet count was low ($82 \times 10^9/L$). Although not confirmed, these clinical and complementary findings were suggestive of HPS; a condition that has been described in the Brazilian Central Plateau either isolated or as a coinfection.³⁻⁵ On a practical standpoint, corticosteroids have been useful to control both the syndromes.^{1,5}

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